The Public Health Impact of Socioeconomic Status on Adolescent Depression and Obesity

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Objectives. We examined the public health impact of the socioeconomic status (SES) gradient on adolescents' physical and mental health.

Methods. Population attributable risk (PAR) for household income and parental education were calculated relative to depression and obesity among a nationally representative sample of 15 112 adolescents.

Results. PARs for income and education were large. Across each gender and race/ethnicity group, the PAR for education tended to exceed that for income. For depression, the adjusted PAR for income was 26%, and the PAR for education was 40%; for obesity, the adjusted PAR for income was 32%, and the PAR for education was 39%.

Conclusions. SES is associated with a large proportion of the disease burden within the total population. (*Am J Public Health.* 2003;93:1844–1850)

Understanding the impact of social inequalities on health has become a public health priority in the new millennium.[1] Social, political, and economic factors now are acknowledged to be "fundamental" causes of disease that affect behavior, beliefs, and biology.[2] This recognition is changing the theoretical framework of epidemiology by incorporating the complex, interactive processes that create population health differentials.[3] Understanding this sociobiological translation among adolescents is critically important, because adolescence is the time of transition between family-determined social status of childhood and adult social status, which is largely self-determined.[4] Throughout industrialized countries, lower adult socioeconomic status (SES) has been clearly linked to poorer health.[5,6] Additionally, SES gradients in adolescent health have been documented in both the United States and Europe.[7–9]

Despite the pervasive nature of the SES–health relationship and the importance of adolescence in setting the trajectory for adult health, few studies have assessed the SES–adolescent health gradient.[10] These studies have led to conflicting views on the importance of SES and other social factors, such as race/ethnicity and family structure, in creating health differentials.[11,12] Some investigators have concluded that these social factors should be discarded as useful mechanisms for understanding adolescent health differentials.[11,13] However, the analyses on which these conclusions were based, such as regression analyses, focus on predicting interindividual risks.[14] They do not consider the broader population-level effects of SES on adolescent health.

Population attributable risk (PAR) is a concept that has been developed to determine the population-level or public health impact of an exposure on an outcome.[15] First described in 1953 by Levin,[16] PAR represents the proportion of cases of a disease that would be prevented if the risk factor or the exposure were removed from the population. Although Levin's definition was for a dichotomous exposure variable, the concept of attributable risk has been extended to polytomous exposure variables, such as SES, and methods have been developed to adjust for other related factors.[17]

To investigate the population-level impact of SES on adolescent health, we used data from the National Longitudinal Study of Adolescent Health (Add Health),[18] a nationally representative sample of youths in grades 7 through 12, to determine the PAR due to lower education and lower household income relative to adolescents' physical and mental health. We hypothesized that, despite their modest predictive performance at the individual level, lower education and lower household income would have substantial population-level effects on 2 major public health problems of youth: depression and obesity. Both of these morbidities are linearly associated with SES among teenagers, and both are important and increasing public health problems.[7]

METHODS

Sample

Data for this study were drawn from the Wave 1 in-home weighted sample of Add Health. There were 18 922 subjects who were assigned a grand sample weight in the Wave 1 in-home sample.[19] Of these, 82% (n=15 484) had a parent complete the parental interview. All of the subjects for whom a parent answered questions that assessed parental education, household income, or both were included in analyses (97.6%, n=15 112). There were no significant differences in age, gender, or race/ethnicity between those whose parent answered at least 1 of the SES-related questions and those whose parent did not.

SES Indicators

Measures of SES were drawn from information obtained during the parental rather than the adolescent interview. Parental reports of overall 1994 household income were categorized into quintiles according to 1994 US Census data for household incomes.[20] Parental respondents also reported educational attainment for self and current spouse or partner. The higher of these was used to create a 5-level ordinal variable as described in previous analyses that used Add Health data.[7,21] Categories included less than high school; high school degree, general equivalency degree (GED), or vocational training instead of high school; vocational training after high school or some college; college graduate; and professional training beyond college.

Health Outcomes

Obesity. Body mass index (BMI, kg/m^2) was calculated from adolescents' self-reported height and weight. BMI *z* scores and percentiles then were determined on the basis of Centers for Disease Control and Prevention (CDC) revised growth charts.[22] Obesity was defined as a BMI greater than or equal to the 95th percentile for age and gender.[23] Use of self-reported height and weight to calculate BMI has been validated among youths.[24] Although measured height and weight were available for wave 2 of Add Health, analyses with baseline data are reported because there was significant attrition in the follow-up sample, and parallel analyses that examined measured BMI in the follow-up cohort yielded virtually identical results.

Depression. We used a well-validated and widely used epidemiological survey tool—the Centers for Epidemiologic Study–Depression Scale (CES-D)—to assess depressive symptoms.[25,26] The CES-D has been widely used in studies of adolescents' emotional health.[26–29] Roberts et al. used the receiver operating characteristic (ROC) curve to analyze data obtained from a large, diverse community sample of students in grades 9 through 12. They determined that scores of 24 or greater for females and 22 or greater for males maximize the sensitivity and the specificity of the CES-D for predicting major depressive disorder as defined by *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (*DSM-III*), criteria.[27] A dichotomous variable that indicates depression was created on the basis of these cutpoints.

Covariates. Sociodemographic covariates included age, gender, and race/ethnicity. The sample was 56.7% non-Hispanic White, 19.8% non-Hispanic Black, 16.5% Hispanic, 5.3% Asian, and 1.7% other race/ethnicity. For analytic purposes, this variable was dichotomized to non-Hispanic White versus other.

Analytic Strategy

Mathematically, PAR can be defined as follows:

$$1)\quad \frac{PAR = P(D/E)P(E) - P(D/\hat{}E)P(E)}{P(D)}$$

where P(D)=probability of disease, P(E)=probability of exposure, P(^E)=probability of nonexposure, P(D/E)=probability of disease given exposure, and P(D/^E)=probability of disease given nonexposure.[30(p76)]

PAR represents the proportion of disease that would be prevented if the exposure were removed and if the entire population achieved the disease prevalence in the previously unexposed group.[30] In our study, the SES gradient defined the current exposure pattern. We performed 2 estimates of PAR. The first estimate addressed SES effects throughout the gradient. To do this, we defined the unexposed category as those in the top income quintile for household income and as those with a professional degree beyond college for parental education. PAR derived from this definition of exposure assessed what proportion of depression and obesity among adolescents would be prevented if all individuals were at the same level of risk as those in the top income quintile or those from families with at least 1 parent who received professional training beyond college. Second, to assess effects of SES among the most vulnerable, and because much of the literature has dichotomized SES as poor versus nonpoor, we determined PAR when the exposed population was defined as those in the lowest income quintile for income and as those who had not graduated from high school or obtained a GED for education.

This set of analyses provided an assessment of PAR due to poverty or lack of a high school–level education. The unexposed population in our analyses included the other 4 categories of income and education collapsed into 1 group. Thus, in the second set of analyses, PAR assessed the proportion of disease that would be prevented if the most vulnerable were given a risk equivalent to the average level of risk among the rest of the population. In addition to PAR, we calculated attributable risk among the exposed (AR_e) for this dichotomization. Attributable risk among the exposed is a calculation of the proportion of cases that are due to the specific risk factors of interest within the exposed population—those in the lowest income quintile or those without a high school education. It is not a populationwide measure. This statistic shows the importance of risk factors in determining prevalence within the most disadvantaged groups.

Before determining either PAR or AR_e, we verified the association between each SES indicator and the 2 health outcomes, in the total population and 4 strata—white males, white females, non-white males, and non-white females. For each stratum, a 5-by-2 (SES × health outcome) table was created. We used the Cochran-Armitage Trend test to test for the linear association between SES and the outcome of interest in each stratum in order to assess the SES gradient effect. Additionally, we used the Cochran-Mantel-Haenszel statistic to test for the overall association adjusted for the strata. Once the above tests established the SES–health association ($P<.05$), the adjusted PAR was calculated across the entire SES gradient according to Bruzzi's method.[31]

All analyses were conducted with SAS v8.01 software (SAS Institute, Cary, NC), and sample weights were used to adjust for the differential probability of selection. All statistical significance testing was performed with SUDAAN v8.0 software (Research Triangle Institute, Research Triangle Park, NC) to account for the complex cluster design of Add Health. Unadjusted PAR was first calculated for the entire population and then stratified by gender multiplied by race/ethnicity to account for the covariance of gender and race/ethnicity, because important racial and gender differences existed for both these outcomes.

RESULTS

Sample

Mean age of the 15 112 students was 16.1 ± 1.7 years. The sample was 48.8% female and 69.6% non-Hispanic White. Household income was missing for 10.1%; when compared with those who had income data, the students who did not have income data were more likely to be non-White ($P<.001$) and to not have a parent in the top education category ($P=.03$). In our study, 12.6% of the students lived in households in the lowest income quintile, 15.6% were in the second income quintile, 20.5% were in the third income quintile, 22.1% were in the fourth quintile, and 18.5% were in the top income quintile.

TABLE 1—Prevalence of Adolescent Depression and Obesity Among Adolescents in Wave I of Add Health

	Total Population	White Female	Non-White Female	White Male	Non-White Male
Unweighted N	15,112	4285	3338	4247	3171
Percentage with disease in total population, No. (%)					
Depression	1510 (9.2)	417 (9.5)	456 (12.7)	303 (7.0)	327 (10.0)
Obesity	1543 (10.1)	279 (6.7)	342 (11.5)	477 (11.9)	439 (13.8)
Percentage with disease in top-income-quintile households, No. (%)					
Depression	216 (6.5)	67 (6.6)	47 (10.9)	68 (5.3)	32 (7.0)
Obesity	198 (6.7)	38 (4.3)	31 (6.8)	85 (8.2)	44 (11.6)
Percentage with disease and at least 1 professionally educated parent, No. (%)					
Depression	141 (5.2)	41 (5.6)	34 (7.8)	39 (3.4)	26 (7.1)
Obesity	129 (6.0)	22 (3.6)	23 (6.6)	47 (6.7)	37 (11.8)

Note. Add Health = National Longitudinal Study of Adolescent Health.

Parental education data were missing for 5.3%; when compared with those who had education data, the students who did not were more likely to be male ($P=.03$), non-White ($P=.03$), and in the lower 4 income quintiles ($P=.001$). In our study, 9.6% of the students did not have a parent with a high school degree, 25.6% had a parent with a high school degree or GED, 29.8% had a parent with some college or vocational training beyond high school, 16.3% had a parent who was a college graduate, and 13.4% had a parent with professional training beyond college.

Table 1 shows the prevalence of depression and obesity among these adolescents. Percentages for the total population and for those within the top (unexposed) category for each SES indicator are given. Overall, 10.1% of the total population was obese, and 9.2% was depressed. Although these percentages are nearly identical, there was no significant association between obesity and depression within this population. There also were no differences in the prevalence of depression or obesity among those missing either SES indicator.

Relative Risks

The relative risks for lower SES relative to adolescent depression and obesity are shown in Table 2 for income and in Table 3 for education. Tests for a general association between both SES indicators and both outcomes were significant among all strata ($P<.001$). Most of the tests for a linear effect also were significant, which indicates that, in general, a graded, stepwise relationship exists between both SES indicators and these health outcomes. No graded effect was seen for obesity among non-White males, for either SES indicator, and there was no graded relationship between income and depression among non-White females. Whereas the tests for significance yielded significant P values, the relative risks associated with them were modest: most were well below 2.00 among all strata.

PARs

Unadjusted and adjusted attributable risks are shown in Table 4. In contrast to the moderate values of the relative risks found in Tables 2 and 3, the attributable risks in Table 4 indicate that lower SES produces large attributable risk estimates. By and large, these PARs are between 30% and 50%. For depression the adjusted PAR for lower income was 26% and the adjusted PAR for lower parental education was 40%. The adjusted PARs were 32% for lower income and 39% for lower parental education relative to obesity. PARs were reduced when a graded relationship did not exist between the SES indicator and the health outcome. The lowest PARs were found for income–depression among non-White females (13%), for education–obesity among non-White males (15%), and for income–obesity among non-White males (17%). The highest PARs were found for education–depression among White males (50%), for education–obesity among the total population (50%), and for education–obesity among White females (47%).

When we dichotomized SES into those at the bottom of the SES gradient compared with all others, PARs were lower (Table 4). For depression, the PAR was 7.4% for income and 8.0% for education; for obesity, the PAR was 4.8% for income and 3.2% for education. However, the attributable risk among the exposed—those at the bottom of the SES gradient—was much greater. For depression, the AR_e was 36.4% for income and 50.0% for education; for obesity, the AR_e was 27.0% for income and 25.0% for education.

DISCUSSION

"Case-centered epidemiology identifies individual susceptibility, but it may fail to identify the underlying causes of incidence."[32(p38)]

We used PARs to assess the public health impact of SES on indicators of adolescents' physical and emotional health. Our study shows that SES has a broad and an important influence on health across the population. Overall, lower household income and lower parental education each were associated with approximately one third of depression and obesity in this national sample. A graded relationship between SES and health at the individual level was associated with a higher population-level effect. Thus, these data indicate that SES accounts for a large proportion of the disease burden within the whole population.

PAR is the most commonly used statistical measure for assessing the importance of a risk factor across a population because it is a function of both the relative risk of exposure to that factor and the prevalence of exposure within the population. A factor with a relatively low relative risk and, therefore, low predictive power on an individual level may have significant public health consequences if highly prevalent.[33] The PAR associated with such a factor would be high even though the

TABLE 2—Relative Risk of Adolescent Depression and Obesity Associated With Decreasing Household Income Quintile

	Depression Relative Risk (95% CI)						Obesity Relative Risk (95% CI)					
	Q5[a]	Q4	Q3	Q2	Q1	*P**	Q5[a]	Q4	Q3	Q2	Q1	*P**
Total population	1	1.21 (1.36, 1.95)	1.46 (1.23, 1.74)	1.63 (1.36, 1.95)	2.07 (1.73, 2.47)	< .001	1	1.33 (1.71, 2.44)	1.88 (1.59, 2.22)	1.79 (1.49, 2.12)	2.04 (1.71, 2.44)	< .001
Stratified by gender × race/ethnicity												
White female	1	1.13 (0.85, 1.51)	1.72 1.31, 2.56)	1.77 (1.32, 2.37)	2.8 (2.09, 3.75)	< .001	1	1.3 (0.91, 1.85)	1.96 (1.40, 2.75)	2.08 (1.45, 2.98)	2.72 (1.86, 3.99)	< .001
Non-White female	1	1.04 0.64, 1.71)	1.21 (0.77, 1.90)	0.96 (0.61, 1.51)	1.42 (0.92, 2.17)	NS	1	1.73 (1.09, 1.75)	1.78 (1.01, 3.13)	1.29 (0.72, 2.30)	2.1 (1.21, 3.62)	.04
White male	1	1.43 (1.06, 1.92)	1.33 (0.97, 1.81)	1.73 (1.24, 2.41)	1.73 (1.20, 2.50)	< .001	1	1.38 (0.95, 3.14)	1.83 (1.45, 2.30)	1.66 (1.27, 2.17)	1.82 (1.37, 2.42)	< .001
Non-White male	1	1.06 (0.57, 1.97)	1.17 (0.65, 2.09)	1.57 (0.91, 2.72)	1.57 (0.91, 2.73)	.01	1	0.79 (0.48, 1.31)	1.4 (0.91, 2.15)	1.43 0.94, 2.17)	1.15 (0.74, 1.78)	NS

Note. NS = not significant.
*Test for linear trend; all tests for general association were statistically significant ($P < .001$).
[a]Reference group = unexposed category. Relative risk set to 1 for calculation of population attributable risk (PAR); see Table 1 for prevalence of disease in this group.

TABLE 3—Relative Risk of Adolescent Depression and Obesity Associated With Decreasing Parental Education

	Depression Relative Risk (95% CI)						Obesity Relative Risk (95% CI)					
	Professional Degree[a]	College Graduate	> High School, < College	High School	< High School	*P**	Professional Degree[a]	College Graduate	> High School, < College	High School	< High School	*P**
Total population	1	1.5 (1.10, 1.88)	1.6 (1.31, 1.98)	1.95 (1.59, 2.40)	2.95 (2.37, 3.67)	< .001	1	1.39 (1.12, 1.93)	1.76 (1.45, 2.14)	2.17 (1.80, 2.64)	2.26 (1.82, 2.81)	< .001
Stratified by gender × race/ethnicity												
White female	1	1.22 (0.85, 1.76)	1.93 (1.42, 2.64)	1.87 (1.36, 2.57)	2.55 (1.70, 3.82)	< .001	1	1.47 (0.94, 2.31)	1.81 (1.21, 2.70)	2.66 (1.79, 3.94)	3.08 (1.86, 3.45)	< .001
Non-White female	1	1.75 (1.00, 3.06)	1.19 (0.70, 2.03)	1.79 (1.08, 2.97)	2.08 (1.24, 3.46)	.001	1	1.38 (0.73, 2.68)	1.65 (0.93, 2.94)	2.1 (1.20, 3.69)	1.83 (1.03, 3.29)	.01
White male	1	2.15 (1.41, 3.28)	1.79 (1.20, 2.69)	2.3 (1.54, 3.44)	3.89 (2.44, 6.21)	< .001	1	1.33 (0.96, 1.83)	1.9 (1.44, 2.53)	2.31 (0.74, 3.06)	2.24 (1.54, 3.27)	< .001
Non-White male	1	0.94 (0.49, 1.79)	1.06 (0.61, 1.87)	1.47 (0.85, 2.53)	2.3 (1.35, 3.93)	< .001	1	1.19 (0.75, 1.90)	1.19 (0.78, 1.81)	1.14 (0.74, 1.76)	1.28 (0.83, 1.98)	NS

Note. *Test for linear trend; all tests for general association were statistically significant ($P < .001$).
[a]Reference group = unexposed category. Relative risk set to 1 for calculation of population attributable risk (PAR); see Table 1 for prevalence of disease in this group.

relative risk was low. Our findings show that lower SES represents 1 such factor relative to adolescent health. Lower SES is highly prevalent among America's youth. Almost two thirds of adolescents live in homes without a college-educated parent, and almost half live in households with incomes below 2.5 times the federal poverty threshold.[7] Additionally, socioeconomic inequality is increasing in the United States, which suggests that exposure to lower SES among teenagers will increase in coming years.[34]

The patterning of PARs shown here is noteworthy. In general, the PAR for lower parental education was higher than the corresponding PAR for lower household income. Why the PAR for education is larger than the PAR for income is not clear. One factor may be that more individuals fell into the exposed category relative to education (87%) than relative to income (80%). Another factor may reflect the fact that SES is multifaceted. Separate components of SES, such as income and education, may act through different pathways to produce health differentials.[1,21,35] For example, education's effect may relate more to differences in coping styles and other interpersonal skills, such as communication, whereas income's effect may be more strongly associated with material goods and services. We calculated PAR separately for income and education because of these potential differences in underlying mechanisms and because income and education are not synonymous. For example, in our study population, only 38% of adolescents who lived in households in the top income quintile had a parent with a professional degree beyond college. We also showed that the lowest PARs were found in strata where no graded relationship was present between the SES indicator and the health outcome. This suggests that a graded linear relationship between SES and health outcome may be more detrimental to health than a nonlinear association. Additionally, the highest PARs were found among strata with the steepest SES gradients, which suggests that the steeper the gradient, the worse the population health effects.[36]

Studying population-level effects of SES requires researchers to move beyond a focus on poverty in order to understand social inequalities in health.[1,6] Many studies of ado-

TABLE 4—Attributable Risk of Household Income and Parental Education Relative to Adolescent Depression and Obesity

	Depression		Obesity	
	Income	Education	Income	Education
Unadjusted PARs				
Total population	0.30	0.42	0.43	0.50
Stratified by gender × race/ethnicity				
White female	0.32	0.40	0.38	0.47
Non-White female	0.13	0.38	0.39	0.42
White male	0.27	0.50	0.32	0.44
Non-White male	0.25	0.29	0.17	0.15
Adjusted PARs[a]				
Ordinal income and education	0.26	0.40	0.32	0.39
Income and education dichotomized	0.074	0.08	0.048	0.032
Adjusted Attributable risk in the exposed with income and education dichotomized	0.36	0.50	0.27	0.25

Note. PAR = population attributable risk.
[a]Adjusted for gender and race/ethnicity.

lescent health have focused on poverty and, therefore, have dichotomized SES.[37–43] Although a focus on poverty does not allow exploration of the full range of SES effects on health, it does concentrate on those individuals at greatest risk. We found that when SES was dichotomized and was focused on those at the lowest end of the SES gradient, the attributable risk in the exposed (those in the lowest income quintile or those without a high school degree) was profound. This suggests that policies that focus on eliminating poverty or ensuring a high school education or its equivalent for all can have important effects among the most vulnerable. However, to fully understand how SES affects health, research must move beyond a bivariate approach. In our study, the analyses that dichotomized SES revealed much lower overall PARs than the analyses that assessed PAR across the entire SES spectrum. This suggests that policies that focus on the most vulnerable will not change the adverse health effects of SES for most individuals in the population. Although it is not possible to bring all individuals into the highest income quintile or to ensure a professional degree for all US citizens, our study highlights the importance of exploring realistic policy options that could be applied throughout the SES spectrum.

Our study used depressive symptoms as an indicator of adolescents' emotional health and obesity as an indicator of adolescents' physical health. These diseases represent critical, highly prevalent public health problems for today's youth, because both are chronic diseases that track into adulthood.[44,45] Both diseases increase risk for other morbidities and mortality, and they can lead to impaired social, work, and family functioning.[44,46,47] Additionally, both diseases increase risk for cardiovascular disease, the leading cause of death in the United States. Thus, these data indicate that, in addition to its important role in setting the trajectory for major adult health problems, adolescence may be a critical period for determining the well-established SES gradient in adult cardiovascular disease.

Our study used chronic illness rather than particular health risk behaviors to define health, a strategy that was deliberate and somewhat unusual. Adolescents are generally considered a healthy population. Perhaps for this reason, much of the research on adolescent health has focused on adolescent health risk behaviors, such as substance use and sexual health risk behaviors. However, serious psychological and physiological diseases, such as depression and obesity, exist within this age group. The literature's focus on adolescent health risk behaviors has led to the characterization of adolescent health as behavioral health. Behavioral health, in turn, is characterized as individually determined by faulty lifestyle choices—adolescents choose to engage in sexual intercourse, smoke cigarettes, drink alcohol, consume high-fat diets, and avoid exercise. Choice is assumed to be the property of the individual, which leads to the assumption that risk is a property of the individual. Yet, behavioral choices are constrained and are determined by socially and biologically mediated processes. In the adolescent health literature, the environmental determinants of choice, such as SES, are often ignored or are viewed as confounders. This perpetuates a "blame the victim" mentality.[48] These data, which take a population perspective rather than an individual perspective, indicate that SES is and should continue to be a critically important public health focus for research and intervention. These data also indicate that to understand youth health and behaviors, the context in which youth live must be considered.

Although PAR is the most commonly used method for assessing the public health impact of a particular risk, there are some limitations to this method that must be acknowledged. To calculate PAR, subgroups must be defined as exposed and unexposed. The use of a broad definition of exposure in determining attributable risk is recommended, as is the use of attainable cutpoints to define unexposed subgroups.[49,50] Whereas the definition of unexposed is often relatively straightforward, the definition of unexposed relative to the SES gradient is complex. Social stratification is an integral part of any organized social group.[51] Social hierarchies exist in any society; thus, the SES gradient will never be eliminated and even within the unexposed, a hierarchy is present. Therefore, the cutpoint used to determine unexposed is an arbitrary one. The unexposed categories in our study were broad categories derived from distribution of economic resources and educational certification. As our data on dichotomizing SES indicates, other methods of defining the unexposed category may yield different PAR estimates. Additionally, 2 assumptions underlie the calculation of PAR. First, the risk factor is assumed to be causal. Work over the past 2 decades indicates that

this assumption appears to be valid relative to the SES gradient in health for the vast majority of morbidities studied.[1,6] Second, calculation of PAR assumes that changing the distribution of the single risk factor of interest will be possible and independent of other associated risks. This is not possible for SES, which must by nature work though other more proximal factors to create health differentials. Thus, these estimates of PAR may overestimate the impact of SES on these health outcomes.

Because SES works through other more proximal factors to create health differentials, some argue that it is not an important etiological factor in determining adolescents' health.[11,13] This view does not incorporate the fundamental nature of the SES–health association.[2] SES has been shown to continue to create health disparities even in the face of changing patterns of more proximal risk factors.[2] The large PARs we documented make clear the need to incorporate sociostructural determinants of health into the framework for research on adolescent health. Because a focus on proximal interindividual risk factors belies the basic nature of the SES–health relationship, such a focus will fail, in the long run, to reduce social inequalities in health. ■

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Contributors

All authors contributed to the conceptualization of this work. B. Huang performed the analyses. All authors interpreted the data. E. Goodman was primarily responsible for writing the article. G. Slap and B. Huang helped in editing and revising the manuscript.

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Human Participant Protection

As secondary analysis of existing data, this study was exempt from human subjects review. Use of the Add Health data and appropriate data security was approved by the Cincinnati Children's Hospital institutional review board.

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